

Unexplained Intraoperative Shock during Surgical Fixation of Proximal Humerus. Possibly Teicoplanin Related Reaction

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Keywords

Intraoperative shock, Anaphylaxis, Teicoplanin.

Introduction

The 6th UK National Audit Project (NAP6) reported the calculated incidence of perioperative anaphylaxis as 1:11,752 with subsequent mortality of 3.8%. Antibiotics are responsible for 48% of the incidents followed by NMBAs (25% of incidents) [1]. Interestingly, Co-amoxiclav and teicoplanin together accounted for 90% of the antibiotics identified, where Teicoplanin was used in more than half of the reported cases [1].

Single doses of Teicoplanin plus gentamicin are the standard antimicrobial prophylaxis for many orthopedic surgeries. Teicoplanin belongs to glycopeptide antimicrobials, a group known to have infusion reactions caused by non-immune histamine release related to the dose and rate infused. Although Teicoplanin has been shown not to cause widespread histamine release, it has a reported incidence of IgE-mediated anaphylaxis between 0.1% and 1% [2].

Case Report

We are reporting a probable case of Teicoplanin anaphylaxis who presented only with unexplained profound hypotension and tachycardia. A 71-yr old woman, ASA class 2 due to hypertension, had previous uneventful GA for curettage and has no known allergies. She was scheduled for urgent fixation of left humerus proximal fracture. She had uneventful GA with ET-tube followed by US-guided Interscalene BPB.

At around 15 minutes of Teicoplanin iv. Infusion of 400 mg, profound shock evolved resistant to treatment with iv. Metaraminol and crystalloids infusion. Adrenaline 0.1 mg iv. Given with which the blood pressure picked up to 80/50 then started on Noradrenaline infusion and continued iv. Crystalloids infusion up to 5000 ml. She had the surgery done and was extubated almost four hours after the incident and sent to ICU on Noradrenaline infusion. Serum Mast Cell tryptase (MCT) measured 30 and 240 min after the reaction were 11.4, 5.4 respectively (normal values <11.4 µg/L), and the next day was 2.4 µg/L. She was referred to Allergy clinic for skin prick testing (SPT).

Discussion

Savic et al. established the following criteria to diagnose allergic anaphylaxis [3]:

1. Reaction within 15 min of administration of Teicoplanin.
2. ≥ features of anaphylaxis should present.
3. Positive skin testing or challenge testing.
4. Raised serum mast cell tryptase (MCT).
5. Alternative diagnosis excluded.

The diagnosis of anaphylaxis is definite if meeting all criteria, probable (if meeting criteria 1,2,5, plus 3 or 4), otherwise uncertain. In anaphylaxis MCT level increases and reaches a peak between 15 min and 2 hours with elimination half-life of 90–120 min. The ideal timing to measure serum MCT level is 1–4 hrs. After the suspected anaphylaxis and need to be compared with a baseline level measured the next day. Acute MCT level above 1.2 × baseline + 2 µg/L is indicative of mast cell activation induced by anaphylaxis [4].

In our case, suspected anaphylaxis presented with unexplained shock almost 15 minutes after Teicoplanin infusion that was resistant to treatment with Metaraminol, treated with iv. dose of 0.1 mg Adrenaline, followed by subsequent continuous iv. infusion of Noradrenaline (0.08 mg/ml, at a rate of 5 – 10 ml/hr.) and extensive fluids. As possible causes of shock were excluded, we were left with one possible cause, a distributive shock induced by anaphylaxis. The acute MCT level measured 30 minutes from the incident, compared to the base line measurement on the next day was suggesting mast cell activation.

Unfortunately, diagnosis of Teicoplanin anaphylaxis, based on MCT and skin testing have shown limitations. One study shows that MCT was raised in many but not all cases of Ig E mediated anaphylaxis to Teicoplanin, this is explained by the existence of an immune pathway not involving MCT release and can precipitate anaphylaxis when activated [5]. Additionally, lacking a standard range of concentrations of Teicoplanin used for skin prick testing is associated with false negative results in patients with suspected anaphylaxis, even with suggestive MCT serum levels [3].

Conclusion

Teicoplanin anaphylaxis is sometimes difficult to prove. It might happen with absence of considerable MCT rise where activation of alternative immune pathway that avoids mast cell degradation

is thought. Also, negative SPT results in suspected cases are not uncommon. Therefore, severity of clinical reaction following Teicoplanin administration should be considered for diagnosis of anaphylaxis and negative SPT results should be treated with caution for any future encounters.

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